EDISON

Therapix Bioscience

Earnings update

Two new cannabinoid programs

Pharma & biotech

Therapix is expanding its cannabinoid-based clinical pipeline beyond neurological disorders to potentially treat obstructive sleep apnea (OSA), a chronic sleep disorder. The company is investigating the efficacy of THX-OSA01, an oral THC/PEA formulation, for the treatment of OSA in a 30-patient Phase IIa trial. In addition, Therapix is exploring the potential of a cannabinoid composition containing an antibacterial agent for the treatment of infectious diseases in preclinical trials. The company reported that the Phase IIa Tourette's study will read out in H118 and the Phase I for mild cognitive impairment will begin in Q118.

Year end	Revenue ($m)	PBT* ($m)	EPADS* ($)	DPADS ($)	P/E (x)	Yield (%)
12/16	0.0	(1.7)	(1.80)	0.0	N/A	N/A
12/17e	0.0	(4.3)	(1.18)	0.0	N/A	N/A
12/18e	0.0	(7.7)	(2.00)	0.0	N/A	N/A
12/19e	0.0	(12.8)	(3.16)	0.0	N/A	N/A

Note: *PBT and EPADS are normalized, excluding amortization of acquired intangibles, exceptional items and share-based payments.

THX-OSA01: A potential pharmacologic for OSA

Partnered with the Assuta Medical Center in Israel, the Phase IIa program is evaluating the efficacy of THX-OSA01 in 30 patients with confirmed OSA for one month. The primary efficacy endpoint is a significant decrease in the Apnea-Hypopnea Index (AHI), a metric for diagnosing and measuring OSA severity that represents the average number of breathing disturbances per one hour of sleep.

OSA is an unmet medical need

An estimated 5.9 million US adults are diagnosed with OSA and currently there are no pharmacotherapies available for treatment. Primary treatment includes positive airway pressure (PAP) devices, oral appliances, and surgery. However, inadequate compliance and efficacy rates have been reported. There is a significant need for therapeutic agents to reduce morbidity associated with OSA such as daytime fatigue, morning headaches, loud snoring and respiratory related arousals.

THX-150: A cannabinoid antibacterial program

The antibacterial properties of THC and other common cannabinoids have been investigated since the 1970s. Therapix is investigating the potential efficacy of THX-150, a drug candidate composed of THC, PEA and an antibacterial agent for the treatment of infectious diseases in a preclinical program in collaboration with the Weizmann Institute of Science and the Tel Aviv Sourasky Medical Center.

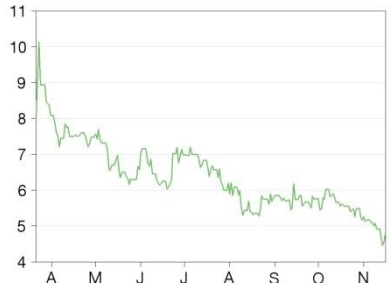
Valuation: Increased to $82.7m or $23.66 per ADS

We have increased our valuation to $82.7m or $23.66 per ADS, from $41.9m or $11.98 per ADS. This change was largely driven by the addition of THX-OSA01 to our model and rolling forward our NPVs, but was mitigated by a lower cash balance. We expect the OSA program to launch in 2022 and achieve peak sales of $754m. Our expected financing requirement for 2018 is $12m with a further $15m to reach profitability in 2021.

17 November 2017

Price	**US$4.84**
Market cap	**US$17m**
	NIS3.49/US$
Net cash ($m) at 30 September 2017	10.7
ADSs in issue	3.5m
Free float	30%
Code	TRPX
Primary exchange	TASE
Secondary exchange	NASDAQ

Share price performance



%	1m	3m	12m
Abs	(12.8)	(10.2)	(39.7)
Rel (local)	(13.7)	(14.3)	(49.2)
52-week high/low	US$10.1		US$4.5

Business description

Therapix Biosciences is an Israeli pharmaceutical company developing cannabinoids for several distinct indications. It is in currently in Phase IIa for Tourette syndrome, entering Phase I for mild cognitive impairment, has initiated Phase IIa for obstructive sleep apnea (OSA), and is beginning preclinical development for infectious diseases and pain.

Next events

THX-TS01 Phase IIa complete	H118
THX-ULD01 Phase I initiation	Q118

Analysts

Maxim Jacobs +1 646 653 7027

Nathaniel Calloway +1 646 653 7036

healthcare@edisongroup.com

Edison profile page

THX-OSA01 for obstructive sleep apnea

Therapix is currently developing THX-OSA01 for the treatment of obstructive sleep apnea (OSA). THX-OSA01 combines tetrahydrocannabinol (THC), the active constituent of cannabis, and palmitoylethanolamide (PEA), a generally regarded as safe (GRAS) lipid with cannabinoid properties. The company recently initiated a Phase IIa program to evaluate the efficacy of THX-OSA01 in 30 patients with confirmed OSA. The primary efficacy endpoint is a significant decrease in AHI after treatment.

OSA is a chronic sleep disorder characterized by recurring episodes of partial (hypopnea: decrease in air flow lasting 10 seconds or more with 30% oxygen reduction) or complete (apnea: cessation of air flow for 10 seconds or more) obstruction of airway passages during sleep causing respiratory-related arousals.[1] OSA occurs when the throat muscles that support the soft palate, uvula, tonsils, side walls of the throat and tongue relax during sleep causing the airway to narrow or close. Signs and symptoms of OSA include daytime fatigue, difficulty concentrating, morning headaches, mood swings and depression, loud snoring, sudden arousals from sleep due to gasping or choking, and high blood pressure. Additionally, OSA is associated with several comorbidities including hypertension, congestive heart failure, obesity and diabetes.

The gold standard diagnostic test for OSA is an overnight polysomnogram, or sleep study, which involves the simultaneous recording of physiologic signals including brain activity, muscle tissue activity, eye movement, breathing patterns and blood-oxygen levels. OSA severity is measured quantitatively using the Apnea-Hypopnea Index (AHI), or the average number of breathing disturbances per one hour of sleep in addition to average oxyhemoglobin desaturation and frequency of arousals from sleep. AHI scores of 0-4, 5-14, 15-30, and >30 correspond to normal, mild, moderate, and severe sleep apnea, respectively.

An estimated 5.9 million[2] adults in the US are diagnosed with OSA while only 54%[3] of those patients diagnosed are actively treated for the condition. Studies suggest that approximately 75%[4] to 80%[5] of severe cases remain undiagnosed, which leads to some variance in estimates of OSA disease prevalence. In addition, the prevalence of OSA is expected to rise along with the ageing population and obesity epidemic. Prevalence estimates from Europe, Australia and Asia are comparable to that of the US.[6]

Treatment options for OSA

Primary treatment options for OSA include positive airway pressure (PAP) devices, oral appliances, and surgery. PAP devices moderately blow pressurized air through the airway at a pressure high enough to keep the throat open and can be delivered through three modes including continuous

[1] Senaratna CV, et al. (2016), Prevalence of Obstructive Sleep Apnea in the general population: A systematic review, *Sleep Medicine Reviews*.

[2] Frost & Sullivan, (2016), American Academy of Sleep Medicine. Hidden health crisis costing America billions: underdiagnosing and undertreating obstructive sleep apnea draining health care system.

[3] Russell, J. O., Gales, J., Bae, C., & Kominsky, A. (2015). Referral Patterns and Positive Airway Pressure Adherence upon Diagnosis of Obstructive Sleep Apnea. *Otolaryngology-Head and Neck Surgery, 153*(5), 881-887.

[4] Maurer, J.T., (2008). Early Diagnosis of sleep related breathing disorders. *GMS Curr Top Otorhinolaryngol Head Neck Surg*.

[5] Frost & Sullivan, (2016).

[6] Punjabi, N. M. (2008). The Epidemiology of Adult Obstructive Sleep Apnea. *Proceedings of the American Thoracic Society, 5*(2), 136-143.

(CPAP), bilevel (BPAP), and autotitrating (APAP).[7] CPAP is the most effective therapy and has shown to improve quality of life; however, approximately 8.2% to 9.2% of newly diagnosed patients refuse CPAP therapy and compliance rates range from 67% to 96%.[8] Oral appliances are recommended to those who are either unable to tolerate CPAP therapy or refuse it. According to the American Sleep Apnea Association, over 100 different devices are FDA approved for the treatment of snoring and OSA. The devices are designed to hold the lower jaw forward to keep the airway open and prevent the tongue and upper airway muscles from collapsing. Efficacy of oral appliance therapy is 52%.[9] Surgical treatment includes a variety of upper airway reconstructive or bypass procedures such as a tonsillectomy and/or adenoidectomy, tongue reduction or stabilization, nasal valve surgery and tracheotomy.[10] Adjunctive to first-line therapies, weight loss, positional therapy and supplemental oxygen are also recommended to relieve symptoms of OSA.

A number of pharmacotherapies have been investigated for OSA management. However, no pharmacologic agent has demonstrated comparable efficacy to PAP. This is a difficult indication to drug because of the multifaceted nature of the neurochemical control and neuromodulation of the central respiratory drive and upper airway motor output.[11] Drugs used to supplement OSA treatment target the reduction or modification of OSA risk factors (nasal congestion, reduced estrogen/testosterone levels), underlying metabolic diseases (thyroid disease, obesity), daytime sleepiness, hypertension and lipid disorders.[12] According to clinical guidelines for the management of OSA in adults, the use of several pharmacotherapies such as methylxanthine derivatives, selective serotonergic reuptake inhibitors (SSRIs), protriptyline and estrogen therapy are generally not recommended for treatment (Exhibit 1). Furthermore, short-acting nasal decongestant sprays do not improve OSA and are not recommended due to concerns with rebound nasal congestion brought about by chronic use. Adjunct topical nasal corticosteroids have been shown to decrease AHI in patients with OSA.[13] There is significant opportunity in this space for a product that can effectively manipulate the respiratory control system to improve OSA.

[7] Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. (2010). Clinical Guideline for the Evaluation, Management and Long-term Care of Obstructive Sleep Apnea in Adults. *Journal of Clinical Sleep Medicine: JCSM: Official Publication of the American Academy of Sleep Medicine*, *5*(3), 263–276.

[8] Abad, V., & Guilleminault, C. (2011). Pharmacological treatment of obstructive sleep apnea. *Current Pharmaceutical Design, 17*, 1418-1425.

[9] Abad, V., & Guilleminault, C. (2011).

[10] Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. (2010).

[11] Prasad, B.et al. (2013). Proof of concept trial of dronabinol in obstructive sleep apnea. *Frontiers in Psychiatry*.

[12] Abad, V., & Guilleminault, C. (2011).

[13] Abad, V., & Guilleminault, C. (2011).

Exhibit 1: Brief summary of ineffective pharmacologic treatment for OSA

Drug	Notes	Recommendation
Methylxanthine derivatives	Stimulate ventilation by blocking adenosine receptors. IV aminophylline and theophylline studies showed no significant change in AHI, worsening of sleep quality and efficiency.	No.
Opioid antagonists	Stimulate ventilation by blocking endorphins that inhibit respiration or stimulating the cortex. Naloxone study did not improve AHI, minimally reduced oxygen desaturation index, and decreased total sleep time.	No.
Doxapram	Stimulates chemoreceptors of carotid arteries to stimulate respiration. Doxapram study improved blood-oxygen desaturation, decreased apnea length, but did not decrease the frequency of apneas.	No.
Nicotinic agents	Nicotinic acetylcholine receptor modulates excitatory inputs to hypoglossal motor neurons (extrinsic and intrinsic muscles of the tongue) and increases diaphragm activity. Studies have shown reduced sleep efficiency, impaired sleep, and variable AHI reduction.	No.
Carbonic anhydrase inhibitor	Stimulates ventilation by inducing metabolic acidosis. Acetazolamide studies showed decreases in AHI, excessive daytime fatigue, and frequent side effects such as burning/tingling sensations, ringing in the ears, nausea, etc.	No.
Paroxetine	SSRI agent to regulate upper airway dilator muscles. Studies showed reduction in AHI during NREM sleep, but not during REM sleep. No improvement in daytime fatigue, morning headaches, depression, or concentration.	No.
Protriptyline	TCA to reduce apnea episodes and increase daytime alertness. Studies have shown mixed results. Two studies showed no AHI or blood-oxygen level improvement. Another study showed reduced AHI during NREM sleep, improved oxygen desaturation and anticholinergic side effects.	No.
Clonidine	Stimulates alpha-adrenoceptors, which reduces sympathetic outflow from the central nervous system, decreases peripheral resistance, renal vascular resistance, heart rate and blood pressure. Studies showed complete REM sleep suppression and reduced time spent in REM sleep. In studies with OSA patients undergoing surgery, an opioid sparing effect was found.	Not for primary treatment, potential for perioperative management.
Mirtazapine	Enhances central respiratory drive by the de-inhibition of the vagal nucleus solitary input. Studies have shown mirtazapine increases sleep continuity and reduces AHI. Side effects include weight gain and increased sedation effects.	No.
Physostigmine	Increases sympathetic autonomic activity. Study demonstrated reduction in AHI during REM sleep. More studies are needed.	Potential for REM-related OSA.
Estrogen and progesterone	Enhance respiratory chemo-sensitivity. Two studies using both estrogen and progesterone showed decreased AHI and shorter hypopneas. In another study, estrogen alone reduced AHI, and progesterone weakened the effects. A progesterone study did not show any AHI improvement. Several risks are associated with hormone replacement therapy including venous thromboembolism, ischemic stroke and breast cancer.	No.
Thyroid replacement	Hypothyroid treatment has ambiguous effects on OSA.	Adjuvant to CPAP.

Source: Adopted from Abad, V., & Guilleminault, C. (2011). Note: SSRI = selective serotonin reuptake inhibitor, NREM = Non-REM, TCA = tricyclic antidepressant.

THC as a vagal afferent modulator for OSA

Several studies have highlighted the role of endocannabinoids as a neuromodulator of cardio-respiratory functions as well as interactions with neurotransmitters related to sleep-wake behaviors. Comparable factors have been shown in animal studies demonstrating respiratory stability improvement with cannabinoid agonists.[14] Increased activity of vagus nerves, peripheral components of respiratory control including respiratory frequency, reduces upper airway activation or openness and therefore may predispose an individual to OSA. The nodose ganglia of the vagus nerves contain receptors for neurochemicals that can modulate vagal afferent activity. Studies have shown that vagal afferent nerves are inhibited by the injection of dronabinol, a non-selective cannabinoid (CB) 1 and CB2 agonist, into the nodose ganglia and the attenuation of nerve activity causes an increase in upper airway activity.[15]

Two small-scale, randomized controlled clinical studies have been performed by RespireRx Pharmaceuticals examining THC for the treatment of obstructive sleep apnea. The company licensed exclusive worldwide rights to develop and commercialize cannabinoids for the treatment of breathing-related sleep disorders from the University of Illinois at Chicago. The first study was a 21-day randomized, placebo controlled, dose escalation (2.5mg, 5mg, 10mg) Phase IIa trial of dronabinol (generic THC) in 22 patients with OSA and it showed a significant decrease (32%) in AHI (events/hour) compared to baseline. 2.5mg (p= 0.007) and 10mg (p=0.036) doses of

[14] Prasad, B.et al. (2013).

[15] Calik, M.W., & Carley, D.W. (2014) Cannabinoid type 1 and type 2 receptor antagonists prevent attenuation of serotonin-induced reflex apneas by dronabinol in sprague-dawley rats. *PLoS ONE*, 9(10).

dronabinol significantly reduced AHI scores compared to baseline. The second study examined 56 patients with moderate to severe OSA in placebo and dose escalation cohorts (2.5mg and 10mg of dronabinol per day at night) over six weeks. It reached similar results, showing significant improvement in AHI scores at 2.5mg ($p<0.02$) and 10mg ($p<0.001$) and overall patient satisfaction (10mg, $p<0.02$). THC caused mild side effects including dry mouth, fatigue, headache, increased appetite, dizziness and sleepiness. RespireRx's dronabinol for OSA program is Phase III ready.

Unlike previous attempts at targeting OSA using THC, Therapix's THX-OSA01 is a co-formulation of THC with PEA, a lipid amide present in food that shares significant structural similarity to endocannabinoids. PEA has been approved for use as a health supplement in some parts of Europe and Canada because small-scale clinical studies,[16] as well as case studies,[17] indicate some benefit for the treatment of chronic inflammation and chronic pain, as well as little to no side effects. The Phase IIa trial will investigate doses up to 10mg THC and 800mg PEA. According to the company, the trial will cost approximately $0.5m and it expects a readout by mid-2018. Therapix believes that the "entourage effect" will enable PEA to enhance the potency of THC and improve its formulation's overall efficacy, although this is by no means guaranteed.

In parallel, Therapix entered into a product development agreement with Cure Pharmaceutical, a US drug delivery company, to develop a THC/PEA formulation on Cure's buccal multilayer oral thin film (OTF), called CureFilm, to determine the most effective drug delivery mechanism for the THC/PEA formulation in an effort to increase drug bioavailability and half-life as well as stimulate rapid onset. Cure is developing a combination of 2.5mg of THS with 200mg PEA on its OTF technology.

THX-150: Antibiotic activity of THC

Therapix is developing THX-150, a drug candidate composed of THC, PEA, and an antibacterial agent for the treatment of infectious diseases. In collaboration with the Weizmann Institute of Science and the Tel Aviv Sourasky Medical Center in Israel, the company is initiating a preclinical program to investigate the potential efficacy of THX-150. According to Dr Itamar Shalit, Professor of Pediatrics at Tel Aviv University, early preclinical trials of THX-150 (with gentamicin as the selected antibacterial agent) were more effective in eliminating resistant bacterial strains than antibiotic controls.

Studies dating back to 1975 have demonstrated the antibacterial properties of THC and other common cannabinoids.[18] However, the action of THC on CB2, primarily expressed on cells of immune system, induces T cell and B cell apoptosis and therefore demonstrates immunosuppressive effects.[19] Today, there are significant challenges facing the treatment of infections caused by multidrug-resistant (MDR) bacteria such as methicillin-resistant *Staphylococcus aureus* (MRSA) and drug-resistant mycobacterium tuberculosis XDR-TB. Antibiotic activity is often measured by the minimum inhibitory concentration (MIC) number, which is the lowest concentration (in µg/mL) of an antibiotic that inhibits the growth of a strain of bacteria. MIC is a measure of susceptibility and is used to determine which class of antibiotic will be most effective

[16] Gabrielsson L, et al. (2016) Palmitoylethanolamide for the treatment of pain: pharmacokinetics, safety and efficacy. *Brit. J. Clin. Pharm.* 82, 932-942.

[17] Keppel J M, et al. (2012) Therapeutic utility of palmitoylethanolamide in the treatment of neuropathic pain associated with various pathological conditions: a case series. *J. Pain. Res*. 5, 437–442.

[18] Van Klingeren, B. & Ten Ham, M. (1975) Antibacterial activity of Δ^9-tetrahydrocannabinol and cannabidiol. Antoine van Leeuwenhock 42: 9-12

[19] Lombard, C., et al. (2007). CB2 cannabinoid receptor agonist, JWH-015, triggers apoptosis in immune cells: Potential role for CB2-selective ligands as immunosuppressive agents. *Clinical Immunology*, 122(3), 259-270.

for a particular strain of bacteria. The antibiotic activity of THC compares positively to common antibiotics in several drug-resistant strains of *Staphylococcus aureus* (Exhibit 2) and demonstrates the potential for THC as an antibacterial agent.[20] It is important to note that the reported data are preclinical and that further clinical trials are needed to support the use of THC for this indication.

Exhibit 2: MIC values of THC and antibiotics toward strains of *Staphylococcus aureus*

Compound	SA-1199B	RN-4220	XU212	ATCC25923	EMRSA-15	EMRSA-16
THC	2	1	1	1	2	0.5
norfloxacin	32	1	4	1	0.5	128
erthromycin	0.25	64	128	0.25	128	128
tetracycline	0.25	0.25	128	0.25	0.125	0.125
oxacillin	0.25	0.25	128	0.125	32	128

Source: Adopted from Appendino, G., et al. (2008). Notes: MIC = minimum inhibitory concentration (µg/mL); SA-1199B = fluoroquinolone-resistant strain also characterized as an antibiotic efflux pump; RN-4220 = macrolide-resistant strain; XU212 = tetracycline-resistant strain; ATCC25923 = standard laboratory strain; EMRSA-15 and EMRSA-16 = epidemic MRSA strains occurring in UK hospitals.

THX-TS01 and THX-ULD01 program updates

Therapix announced with its Q317 financial report that the ongoing Phase IIa trial at Yale University examining THX-TS01 for Tourette's is scheduled to complete enrolment by the end of November 2017 and a readout is expected in H118. Seventeen of the total of 18 patients have been enrolled in the study and there have been no reported safety issues to date, which is consistent with the known profile of cannabinoids. The company also provided an update on the planned placebo-controlled, Phase IIb 13-week trial, which will now be conducted as an investigator-sponsored trial at the Hannover Medical School in Germany, instead of in the US, as the company previously stated. Additionally, Therapix expects to submit the Investigational Medicinal Product Dossier for this trial by year-end 2017.

The company also provided updates regarding the mild cognitive impairment program, which remains focused on traumatic brain injury (TBI). Therapix completed the development of the sublingual ultra-low dose formulation (0.4mg of THC) and plans to initiate the Phase I pharmacokinetics (PK) study in Q118. Following the PK study, which will take approximately one month, the company will be initiating a proof-of-concept (POC) study enrolling patients in the acute phase of TBI, which are typically patients as they are admitted to the emergency room. The primary endpoint of the POC study is a measure of cognitive functions post injury.

Valuation

We have increased our valuation to $82.7m or $23.66 per ADS, from $41.9m or $11.98 per ADS. We have added the OSA program to our valuation and determined a risk-adjusted NPV of $40.8m for the program. This is based on a 5% probability of success, which is due to its early stage and lack of clarity on portfolio prioritization, although we will likely increase this as the trial progresses. We assume the target market will be approximately 1.7 million and 2.9 million patients in the US and in Europe, respectively, who are diagnosed with severe OSA and receive treatment for their symptoms. We assume a (WAC) pricing of $3,500 in the US and $2,500 in Europe. The pricing is approximately at parity with the cost of renting a CPAP machine ($250/month). We also assume 30% discounts on the gross price in both territories. COGS are predicted at 13%, which includes an 8% royalty due to Dekel for the THC/PEA combination technology. We forecast that the company will have exclusivity through the 2035 expiration of its patents.

[20] Appendino, G., et al. (2008) Antibacterial cannabinoids from Cannabis sativa: A structure-activity study. Journal of Natural Products, 71(8), 1427-1430.

Other changes to our valuation include lower net cash and advancing our NPVs to the current period. Additionally, the company is working to establish a pain program partnered with Yissum, the tech-transfer company of the Hebrew University of Jerusalem. This opioid-sparing program, which is scheduled to begin during Q417, will evaluate the use of synthetic cannabinoids in combination with opioids in a preclinical rat model to allow for reduced opioid doses without changing analgesic efficacy. We expect to update our valuation with the release of interim data from the THX-TS01 Phase IIa trial expected during H118 and also with the antibacterial program and opioid-sparing program if they enter the clinic, along with any further clarity as to which of these relatively early stage programs will be prioritized.

Exhibit 3: Valuation of Therapix

Development program	Region	Prob. of success	Launch year	Peak sales ($m)	Margin	rNPV ($m)
THX-TS01	US	10%	2021	177	55%	15.49
THX-TS01	Europe	10%	2021	120	55%	15.03
THX-TS01	Development costs					(2.48)
THX-ULD01	US	5%	2022	69	50%	2.80
THX-ULD01	Europe	5%	2022	106	57%	4.69
THX-ULD01	Development costs					(2.02)
THX-OSA01	US	5%	2022	347	55%	21.80
THX-OSA01	Europe	5%	2022	407	49%	21.37
THX-OSA01	Development costs					(1.75)
Unallocated costs						(2.92)
Total						$72.0
Net cash and equivalents (Q317) ($m)						$10.7
Total firm value ($m)						**$82.7**
Total ADS (m)						3.5
Value per ADS ($)						**$23.66**

Source: Edison Investment Research, Therapix reports

Financials

Therapix reported a loss of $1.03m for Q317 attributed to R&D ($0.34m) and SG&A ($0.77m) expenditure. The company expects the current cash balance of $10.7m will be sufficient to fund current operations into Q318. We have increased our SG&A estimates for 2017 and 2018 by around $0.2m each year, due to increased spending on business development. Also, we have increased our R&D estimates by $0.3m in 2017 and $0.6m in 2018 due mainly to the addition of the Phase IIa OSA program. We expect the company to spend a total of $4.4m, including R&D and SG&A, in 2017.

Exhibit 4: Financial summary

$'000s	2016	2017e	2018e	2019e
31-December	IFRS	IFRS	IFRS	IFRS
INCOME STATEMENT				
Revenue	0	0	0	0
Cost of Sales	0	0	0	0
Gross Profit	0	0	0	0
R&D	739	1,413	5,082	10,110
SG&A	1,267	2,939	2,998	3,058
EBITDA	(1,675)	(3,993)	(7,721)	(12,809)
Normalized operating profit	(1,679)	(3,997)	(7,725)	(12,813)
Amortization of acquired intangibles	0	0	0	0
Exceptionals	7	0	0	0
Share-based payments	(327)	(355)	(355)	(355)
Reported operating profit	(1,999)	(4,352)	(8,080)	(13,168)
Net Interest	(6)	(349)	9	13
Joint ventures & associates (post tax)	0	0	0	0
Exceptionals	0	0	0	0
Profit Before Tax (norm)	(1,685)	(4,346)	(7,717)	(12,800)
Profit Before Tax (reported)	(2,005)	(4,701)	(8,072)	(13,155)
Reported tax	0	0	0	0
Profit After Tax (norm)	(1,685)	(4,346)	(7,717)	(12,800)
Profit After Tax (reported)	(2,005)	(4,701)	(8,072)	(13,155)
Minority interests	0	0	0	0
Discontinued operations	0	0	0	0
Net income (normalized)	(1,685)	(4,346)	(7,717)	(12,800)
Net income (reported)	(2,005)	(4,701)	(8,072)	(13,155)
Basic average number of ADS outstanding (m)	1	4	4	4
EPS - normalized (c)	(179.9)	(118.4)	(200.1)	(316.2)
EPADS - diluted normalized ($)	(1.80)	(1.18)	(2.00)	(3.16)
EPADS - basic reported ($)	(2.14)	(1.28)	(2.09)	(3.25)
DPADS (c)	0.00	0.00	0.00	0.00
BALANCE SHEET				
Fixed Assets	441	31	31	31
Intangible Assets	0	0	0	0
Tangible Assets	11	31	31	31
Investments & other	430	0	0	0
Current Assets	804	9,996	14,896	2,937
Stocks	0	0	0	0
Debtors	117	188	0	0
Cash & cash equivalents	676	9,773	14,861	2,902
Other	11	35	35	35
Current Liabilities	(672)	(656)	(1,269)	(2,106)
Creditors	(672)	(656)	(1,269)	(2,106)
Tax and social security	0	0	0	0
Short term borrowings	0	0	0	0
Other	0	0	0	0
Long Term Liabilities	0	0	(12,000)	(12,000)
Long term borrowings	0	0	(12,000)	(12,000)
Other long term liabilities	0	0	0	0
Net Assets	573	9,371	1,658	(11,138)
Minority interests	0	0	0	0
Shareholders' equity	573	9,371	1,658	(11,138)
CASH FLOW				
Op Cash Flow before WC and tax	(1,675)	(3,993)	(7,721)	(12,809)
Working capital	233	(87)	801	836
Exceptional & other	(38)	14	0	0
Tax	0	0	0	0
Net operating cash flow	(1,480)	(4,066)	(6,920)	(11,973)
Capex	(4)	(22)	0	0
Acquisitions/disposals	0	0	0	0
Net interest	0	0	9	13
Equity financing	913	12,900	0	0
Dividends	0	0	0	0
Other	(349)	(22)	0	0
Net Cash Flow	(920)	8,790	(6,912)	(11,960)
Opening net debt/(cash)	(1,596)	(676)	(9,773)	(2,861)
FX	0	307	0	0
Other non-cash movements	0	0	0	0
Closing net debt/(cash)	(676)	(9,773)	(2,861)	9,098

Source: Edison Investment Research, Therapix Biosciences reports

Frankfurt +49 (0)69 78 8076 960
Schumannstrasse 34b
60325 Frankfurt
Germany

London +44 (0)20 3077 5700
280 High Holborn
London, WC1V 7EE
United Kingdom

New York +1 646 653 7026
295 Madison Avenue, 18th Floor
10017, New York
US

Sydney +61 (0)2 8249 8342
Level 12, Office 1205
95 Pitt Street, Sydney
NSW 2000, Australia